

Smoking And Periodontal Diseases

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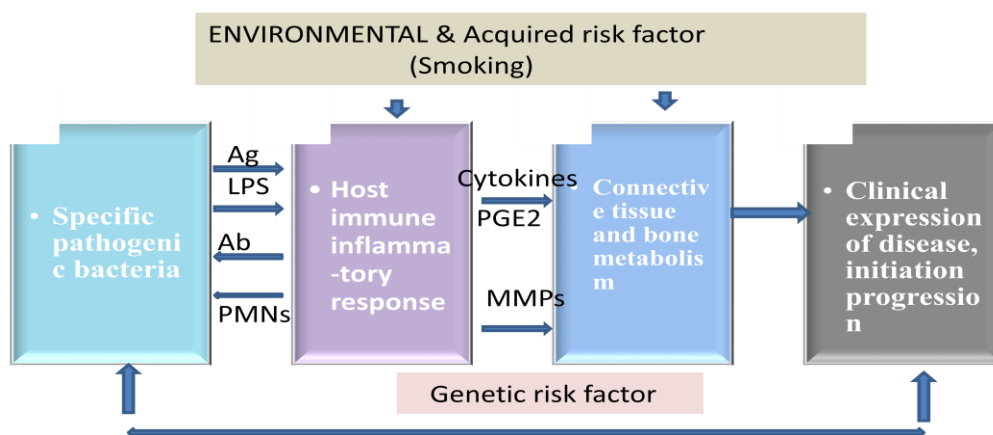
ABSTRACT:

Periodontitis is the result of complex interrelationships between infectious agents and host factors. Environmental, acquired, and genetic risk factors modify the expression of disease and may, therefore, affect the onset or progression of periodontitis. The study of the relationship between periodontal disease and smoking has received increased attention during the last few years. Tobacco smoking has wide spread systemic effects, many of which may provide mechanisms for the increased susceptibility to periodontitis and the poorer response to treatment. Tobacco smoking is a significant risk factor for periodontal disease.

KEY WORDS: Tobacco, smoking, periodontitis.

INTRODUCTION

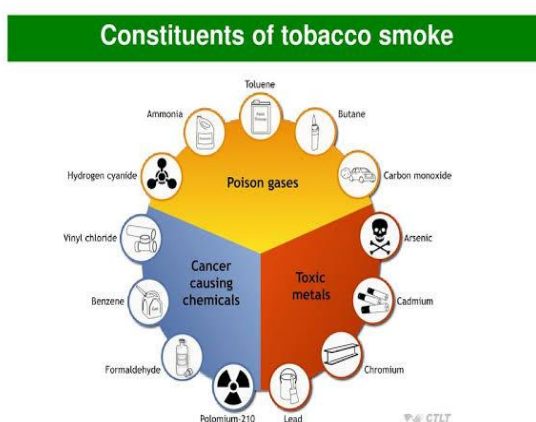
Periodontal disease is defined as an inflammatory destruction of periodontal tissue and alveolar bone supporting the teeth. Progression and the severity of disease depends on complex interactions between several risk factor such as microbial, immunological, environmental and genetic factors, and also age, sex and race. Tobacco smoking is one of the risk factors associated with chronic destructive periodontal tissue. The harmful effects manifest themselves by interfering with the vascular and immunological reaction, as well as by undermining supportive functions of the periodontal tissues. Tobacco is one of the significant risk factors for periodontal disease¹.



Constituent Of Tobacco

Tobacco contains more than 4,000 chemicals, many of which are harmful. These include:

1. **Benzene**- solvent used in fuel manufacture
2. **Formaldehyde**- highly poisonous, colourless liquid used to preserve dead bodies.
3. **Ammonia**- chemical used in cleaning fluids. Used in cigarettes to increase the delivery of nicotine.
4. **Hydrogen cyanide**- poisonous gas used in manufacture of plastics and pesticides.
5. **Cadmium**- extremely poisonous metal found in batteries
6. **Acetone**- solvent found in nail polish remover.



Potent carcinogen:

1. Nitrosamines
2. Polycyclic aromatic hydrocarbons
3. Radiation emitting polonium

Components of inhaled smoke:

1. Nicotine
2. Carbon monoxide
3. Tar

Nicotine Action: Accelerates release of neurotransmitter dopamine in the brain's nucleus accumbens (NA) and increase metabolism in NA. Nicotine retards growth of gingival fibroblasts reduces fibronectin & collagen increases collagen breakdown. They raise blood pressure cause vasoconstriction, Increase respiratory rate, increase heart rate, decrease skin temperature. Nicotine is absorbed slowly in oral mucosa. From cigarette smoke (pH- 5.5) not well absorbed and from cigar & pipe smoke (pH- 8.5) good absorption (Benowitz 1988)

Carbon Monoxide Actions: It is a poisonous gas found in car fumes, which reduces the amount of oxygen carried in the blood. The reduction in oxygen changes the consistency of the blood, making it thicker and putting the heart under increased strain as it pumps blood around the body.

Tar Actions: Tar contains many substances proven to cause cancer. Irritants found in tar damage the lungs causing narrowing of bronchioles and damaging the cilia that protect lungs from dirt & infection.



Different methods of tobacco smoking

Age, Sex And Cigarette Smoking

Carranza stated women from age 20-39 yrs and men from age 30-59 yrs who smoke cigarettes have twice the chance of having periodontal disease or becoming edentulous as do non smokers. The effect of smoking on periodontal status to be more pronounced in younger women².

EFFECT OF SMOKING ON PLAQUE

Effect Of Smoking On Plaque Development

Higher prevalence of dental plaque in smokers. In contrast, smoking did not appear to increase the amount of plaque. Experimental gingivitis study rate of plaque formation was similar³.

Effect Of Smoking On Oral Flora

No significant trend for smokers to harbor putative pathogens. Increased counts of exogenous flora – *E. Coli* and *C. albicans* – have been reported in smokers.

Effect Of Smoking On The Subgingival Microflora

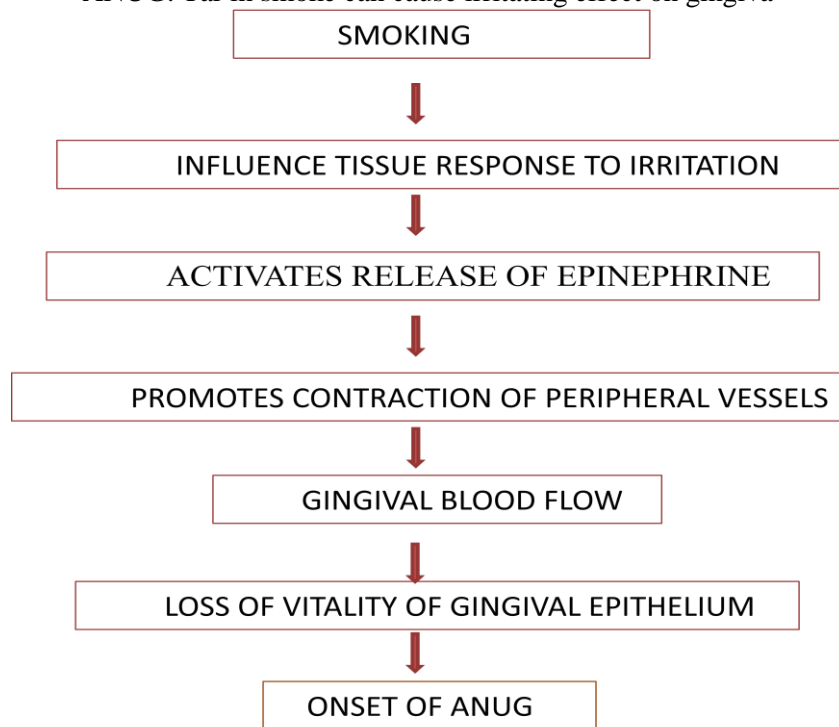
Zambon et al 1996 study showed the proportions of subjects positive for *A. actinomycetemcomitans*, *P. gingivalis*, and *T. forsythesis* were higher among smokers. Prevalence of *B. forsythus* and *P. nigrescens* maxilla > mandible. Cigarette smoking lowers redox potential increase in anaerobic bacteria.

EFFECT OF SMOKING ON PERIODONTAL TISSUE

Effect Of Smoking On Gingiva:

Epithelium changes to hyperkeratotic, hyperplastic and Greyish discolouration of gingiva. Increase amount of IL-1, IL-6 and PGE2 (Johnson et al 1996). Smoking is one of the predisposing factors for

ANUG. Tar in smoke can cause irritating effect on gingiva



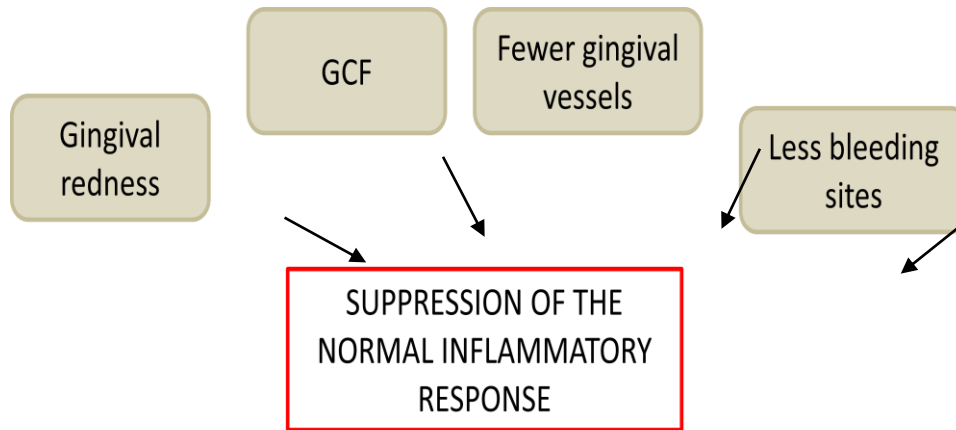
Effect Of Smoking On Gingival Blood Flow:

In smokers, gingival blood flow was significantly increased. However, intravenous administration of nicotine reduces marginal temperature of gingival sites suggesting decrease in gingival blood flow which lead to the phenomenon vasoconstriction induced by nicotine and stress.

Gingival Inflammation And Bleeding

Smokers expressed less gingival bleeding than non smokers. (Bergstrom et al 1983). This may be due to vasoconstrictive effect of nicotine (Clarke et al 1984)

Oxygen tension in the gingival tissue is affected by smoking, oxygen saturation of hemoglobin is affected by cigarette smoking. Oxygen tension was significantly lower in smokers³. On gingival vascularisation smoking cause decrease in number of vessels and decrease in endothelial ICAM-1 expression



Smoking also affects the gingival fibroblast Function. Some studies have shown reduction in the production of Type 1 collagen and fibronectin and an increase in the collagenase activity. Cellular changes like disruption of cell orientation, changes in cytoskeleton, presence of large vacuoles, and significant reduction in cell viability have been noticed⁴.

Periodontal (PDL) fibroblasts growth, attachment and integrin expression was inhibited by nicotine at high concentrations (≥ 1 mg/ml) Nicotine at high concentrations (100 ng/ml to 25 μ g/ml) is cytotoxic by inhibiting the vacuolation and proliferation of fibroblasts³. PDL cell proliferation and protein synthesis were inhibited in a dose dependent manner. Cell attachment was significantly less on root surfaces obtained from heavy smokers compared with nonsmokers and healthy controls³.

Smoking And Periodontitis

Current smokers had deeper probing depths, greater attachment loss, more bone loss, and fewer teeth. Among 20-49 yrs old, the adjusted odds ratio for a mean attachment loss of 1 to 1.99mm among current smokers was 2.29, whereas the odds ratio for attachment loss ≥ 3 mm was over 18.43. This suggests smoking is particularly important in the etiology of severe periodontal attachment loss. (NHANES). There is a strong dose-response relationship between the amount smoked and the severity of periodontal destruction which further supports the role of smoking as a risk factor for periodontitis¹⁰. The most marked difference between smokers and non-smokers in probing depths or attachment loss occurs in the maxillary lingual area and mandibular anterior area, suggesting a local effect of smoking.

Effect Of Smoking On Periodontal Therapy

Several studies demonstrated higher levels of oral debris in smokers than in non-smokers. Increased levels of debris observed in smokers have been tentatively attributed to personality traits leading to decreased oral hygiene habits, increased rates of plaque formation, or a combination of the above⁶.

Non-Surgical And Surgical Therapy

Numerous studies have shown smoking compromises probing depth and/or attachment gain outcomes following non-surgical or surgical therapy. The numerical differences between smokers and non-smokers become more pronounced in probing depths ≥ 5 mm, where smokers demonstrated 0.4 mm to 0.6 mm. Following flap debridement surgery, smokers experienced up to 1 mm less improvement in clinical attachment levels in probing depths that were initially ≥ 7 mm⁷.

Antimicrobial Therapy In Smokers

Because of the diminished treatment response in smokers, clinicians may recommend adjunctive antimicrobial therapy for smokers. Subgingival pathogens are more difficult to eliminate in smokers following scaling and root planning. Systemic amoxicillin and metronidazole or locally delivered minocycline microspheres enhanced the results of mechanical therapy. A recent study reported a positive response to sub-antimicrobial doxycycline (anticollagenase) therapy in combination with scaling and root planning in a group of severe periodontitis patients that included smokers⁷.

Soft And Hard Tissue Grafting

In guided tissue regeneration procedures, smokers had significantly less root coverage (57%) compared to non-smokers (78%). Smoking is detrimental to regenerative therapy in interproximal and furcation defects, whether treatment includes the use of osseous graft, bioabsorbable membrane, or combination. The results from various studies have shown less than 50% as much improvement in clinical attachment levels in smokers compared to nonsmokers, which amounted to differences ranging from 0.35 mm to 2.9 mm⁹.

Implant Therapy

Mostly, 0% to 17% of implants placed in smokers were reported as failures as compared to 2% to 7% in non-smokers. The 3-year data demonstrated 8.9% of implants placed in smokers failed as compared to 6% in individuals who had never smoked or had quit smoking. The majority of implant failures in smoking occurred prior to prosthesis delivery⁸.

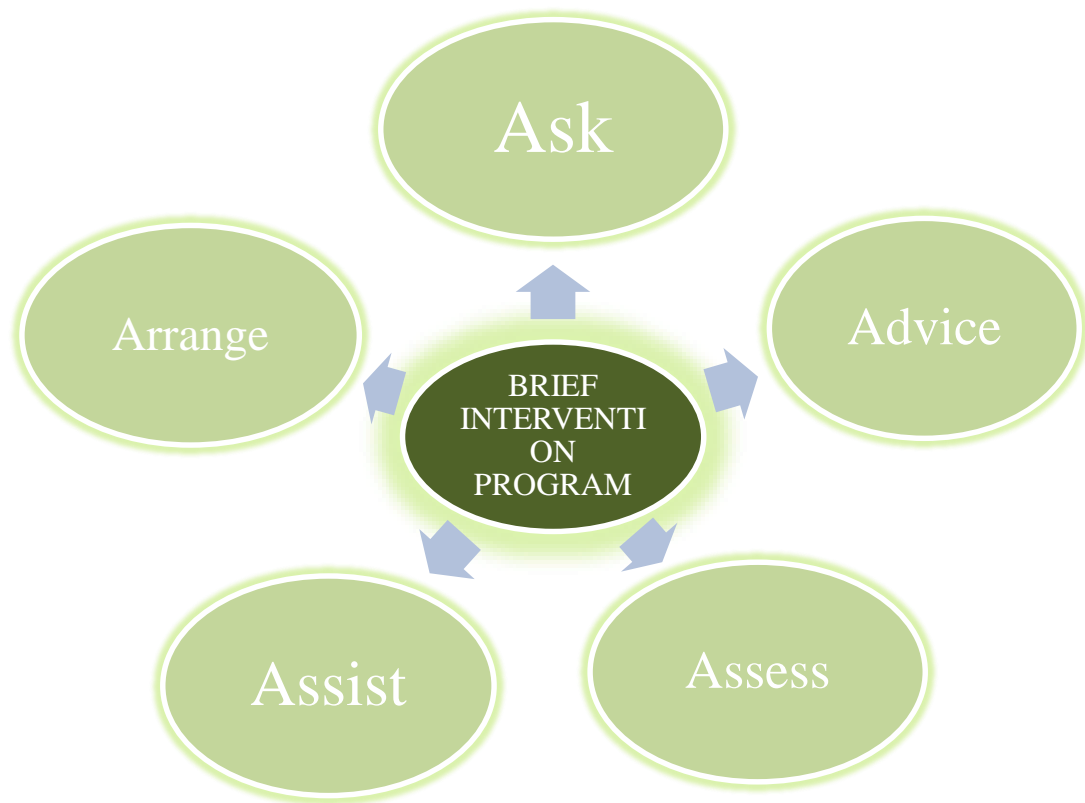
TOBACCO CESSATION

Success of tobacco cessation is based on long term abstinence of about 6 months⁹

BRIEF INTERVENTION PROGRAM

Agency for health care research and quality developed a brief intervention program for tobacco cessation¹¹. This include 5 'A's

1. ASK
2. ADVICE
3. ARRANGE
4. ASSIST
5. ASSESS



Pharmacotherapy

Pharmacotherapy plus behavioural counselling improves long term quit rates. Smokers of 10 or more cigarettes a day who are ready to stop should be encouraged to use pharmacological support as a cessation aid.

Nicotine Replacement:

Begin NRT on the quit date, (apply patches in night)

Use a dose that controls the withdrawal symptoms

NRT provides levels of nicotine well below smoking

Prescribe in blocks of 2 week

Arrange follow-up to provide support

Use a full dose for 6-8 weeks then stop¹.

First-Line Pharmacotherapies for Smoking Cessation (adapted from Fiore et al.¹⁵¹)

Pharmacotherapy	Precautions/ Contra-indications	Side Effects	Dosage	Duration
Bupropion sustained release	History of seizure History of eating disorder	Insomnia, dry mouth	150mg every morning for 3 days, then 150mg twice daily (begin treatment 1 to 2 weeks precessation)	7 to 12 weeks maintenance up to 6 months
Nicotine gum	Temporo-mandibular disorders exacerbated by chewing gum	Mouth soreness, dyspepsia	1 to 24 cigs/day-2 mg gum (up to 24 pieces/day) 25+ cigs/day-4 mg gum (up to 24 pieces/day)	Up to 12 weeks
Nicotine inhaler		Local irritation of mouth and throat	6 to 16 cartridges/day	Up to 6 months
Nicotine nasal spray		Nasal irritation	8 to 40 doses/day	3 to 6 months
Nicotine patch		Local skin reaction, insomnia	22 mg/24 hours 14 mg/24 hours 7 mg/24 hours 15 mg/16 hours	4 weeks then 2 weeks then 2 weeks 8 weeks
Nicotine lozenge		Heartburn, mouth soreness	2 mg lozenge (if first cigarette is 30 min. or more after awaking) 4 mg lozenge (if first cigarette is 30 min. or less after awaking) No more than 20 lozenges per day	Weeks 1 to 6: 1 lozenge every 1 to 2 hours Weeks 7 to 9: 1 lozenge every 2 to 4 hours Weeks 10 to 12: 1 lozenge every 4 to 8 hours

CONCLUSION

Smokers present with periodontitis at an young age and is difficult to treat this patient with conventional therapy. Continued use of tobacco can cause progression and recurrence of periodontitis which further lead to tooth loss. As an environmental factor, smoking interacts with the host and the bacterial challenge, resulting in an increased susceptibility to periodontitis and poorer response to treatment. Recent guidance suggest that dental practices should assess the smoking status of patients and motivate smokers towards quitting. The opportunity for dentists to become more active in evaluation of tobacco use and also can offer counselling to patient on cessation.

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